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Severe burn injury, burn shock, and smoke inhalation injury in small animals. Part 2: diagnosis, therapy, complications, and prognosis

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Abstract

Objective – To review the evaluation and treatment of patients suffering from severe burn injury (SBI), burn shock, and smoke inhalation injury. Potential complications and prognosis associated with SBI are also discussed.
Diagnosis – Diagnosis of burn injury and burn shock is based on patient history and clinical presentation. Superficial burn wounds may not be readily apparent for the first 48 h whereas more severe wounds will be evident at presentation. Patients are diagnosed with local or SBI by estimating total body surface area involved using the 'Rule of Nines' or the Lund-Browder chart adapted from the human literature.
Therapy – Patients suffering from SBI require immediate and aggressive fluid therapy. Burn wounds require prompt cooling to prevent progressive tissue damage. Due to significant pain associated with burn wounds and therapeutic procedures, multimodal analgesia is recommended. Daily wound management including hydrotherapy, topical medications, and early wound excision and grafting is necessary with SBI.
Complications – There are numerous complications associated with SBI. The most common complications include infections, hypothermia, intra-abdominal hypertension, and abdominal compartment syndrome.
Prognosis – The prognosis of SBI in domestic animals is unknown. Based on information derived from human literature, patients with SBI and concomitant smoke inhalation likely have a worse prognosis than those with SBI or smoke inhalation alone.

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Introduction

Severe burn injury (SBI) in small animals has been poorly described in the veterinary literature. Much of the knowledge regarding burn injury in veterinary medicine is derived from advances in human medicine and research animal models. The pathophysiology associated with local burn injury, SBI, and smoke inhalation injury is reviewed on an accompanying article.[1] This review discusses patient evaluation and recommended treatment for animals with SBI and smoke inhalation injury as well as common complications and prognosis associated with SBI and smoke inhalation injury.

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Abbreviations

ABA	American Burn Association
ACS	Abdominal compartment syndrome
ARDS	acute respiratory distress syndrome
SBI	severe burn injury
CM	carbon monoxide
CO-Hgb	carboxyhemoglobin
COP	colloid osmotic pressure
CSA	canine specific albumin
HA	human albumin
HBOT	hyperbaric oxygen therapy
HTS	hypertonic saline
IAH	intra-abdominal hypertension
IAP	intraabdominal pressure
MAP	mean arterial pressure

SpO2	oxygen saturation
SSD	silver sulfadiazine
TBSA	total body surface area
VAC	vacuum-assisted closure

Initial Patient Evaluation

An initial survey evaluation of the SBI patient is required to expedite supportive care and to determine if aggressive medical intervention is necessary. Primary evaluation includes estimating the depth of the burns and percentage of total body surface area (TBSA) involved. The modern burn classification system (see accompanying review[1]) classifies burns by increasing depth.[2–6] The TBSA of human patients is assessed by utilizing the "Rule of Nines" for adults and the Lund-Browder chart for children.[2,3,6–8] Currently, no schematic to estimate TBSA in animals exists and, therefore, caution should be used when these systems are extrapolated for use in small animals. Burn injuries that encompass <20% of the TBSA are referred to as "local burns" whereas those that encompass >20–30% of the body are classified as SBI.[2,9–12] Severe burn injury leads to systemic derangements that require aggressive fluid therapy, multimodal analgesia, and wound management. After the initial survey evaluation is performed, a more thorough examination is essential.

Respiratory System

Severe burn injury with concomitant smoke inhalation injury predisposes the patient to respiratory complications including airway obstruction, hypoventilation, acute respiratory distress syndrome (ARDS), and pneumonia.[11,13–16] A clinical diagnosis of smoke inhalation is made based on the patient's history and physical examination findings.[17,18] Facial burns, singed nasal vibrissae, oropharyngeal blistering, and carbonaceous sputum are all indicators of smoke inhalation in small animals.[6,7,17–21] Bronchoscopy is frequently utilized to aide the diagnosis and predict the severity of inhalation injury in people.[15,22–24] Although this diagnostic modality has proven to be effective and safe in people, its utility in veterinary medicine for the diagnosis of smoke inhalation is undetermined. Due to lack of data and the need for anesthesia in small animals for bronchoscopic evaluation, the authors recommend diagnosing smoke inhalation based on history and physical examination findings alone.

Severe burn injury and smoke inhalation result in significant edema formation and bronchospasm which may compromise airway patency in the early hours after burn injury, therefore, immediate intubation may be necessary.[6,7,13,17,19] Acute airway obstruction occurs in 20–33% of hospitalized human burn patients with smoke inhalation injury.[11] The prevalence of acute airway obstruction in veterinary patients is unknown. Pharyngeal edema resulting in acute airway obstruction typically occurs within the first 24–48 hours after injury and resolves over a few days.[6,7,13,17,19,20] Lower airway and pulmonary complications including sloughing of tracheal membrane and cast formation lead to obstruction of lower airways. This results in segmental atelectasis, which typically occurs 48–72 hours after smoke inhalation.[6,7,17] Patients should be evaluated for hypoventilation secondary to decreased pulmonary and chest wall compliance. Decreased pulmonary compliance can be attributed to bronchospasm, atelectasis, and pulmonary edema. Reduced chest wall compliance can result from torso burns or significant pain.[13] Patients with circumferential or near-circumferential chest wall burns may require an escharotomy to facilitate ventilation.[17]

Dogs and cats presenting immediately after smoke inhalation may appear normal or exhibit only mild increases in respiratory rate and effort. Thoracic auscultation can disclose abnormal lung sounds including increased bronchovesicular sounds, crackles, and wheezes.[25,26] The full effect of smoke inhalation injury to the lungs may not be evident for 24–36 hours after exposure. Initial thoracic radiography may appear normal in these patients.[13,17–21] Radiographs should be re-evaluated based on the progression of clinical illness. Radiographic abnormalities such as a diffuse interstitial pattern, focal alveolar pattern, and lung lobe collapse due to obstruction of main stem bronchi have been reported in dogs and cats after smoke exposure.[25,26]

Cardiovascular System

Severe burn injury, smoke inhalation, and carbon monoxide (CM) toxicity can cause significant cardiovascular abnormalities such as hypotension and cardiac arrhythmias. The patient's cardiovascular system should be assessed by noting mucous membrane color, capillary refill time, cardiac auscultation, and pulse quality. Baseline blood pressure and heart rate should be obtained to guide initial patient resuscitation. If a dysrhythmia is noted, an electrocardiogram should be performed.

Neurologic System

Initial assessment includes evaluation for traumatic injury to the central nervous system. A complete neurologic examination, including cranial nerve evaluation, should be performed. If there is no evidence of head trauma, clinical signs such as agitation, confusion, ataxia,

loss of consciousness, and seizures likely indicate CM or cyanide toxicity.[18,27,28] These clinical signs are likely related to the hypoxic effects on the cerebral cortex. Cellular hypoxia leads to an increase in intracranial pressure and cerebral edema formation.[18,29,30] The nature of clinical signs on initial evaluation may indicate the severity of exposure to both CM and cyanide.[18,28]

Evaluation of both pulse oximetry and arterial blood gas in the face of CO toxicity will yield inaccurate results when evaluating the patient's oxygenation status. A standard 2-wavelength pulse oximeter falsely provides a higher peripheral saturation of oxygen (SpO_2) reading due to its inability to differentiate between oxyhemoglobin and carboxyhemoglobin (CO-Hgb).[5,17,27,30] Arterial blood gas measures the amount of dissolved oxygen in the blood and does not reflect total oxygen content. The partial pressure of oxygen is typically normal in patients with CM toxicity in the absence of parenchymal lung disease.[32] Evaluation of the blood CO-Hgb concentration via a co-oximeter can aide in confirming a diagnosis of CM toxicity.[6,18,28,31–33] Oxygen therapy accelerates CM elimination, and CO-Hgb concentrations may be reduced or normalized after oxygen supplementation. Carbon monoxide toxicity should not be diagnosed solely on the CO-Hgb concentration but also on the patient's history and presenting clinical signs. Patients presenting with neurologic signs, a metabolic lactic acidosis, a normal CoHgb concentration, and normal partial pressure of oxygen likely suffer from cyanide toxicity.[18] Pulse oximetry and arterial blood gas testing should be utilized in patients with smoke inhalation injury and SBI, especially where parenchymal lung injury is a concern, but interpretation of the results must take into consideration these limitations.

Other Body Systems

An ocular examination should be performed shortly after presentation because progressive facial and periorbital swelling may limit visibility of the globe. Patients with facial burns, large TBSA burns, impaired level of consciousness, and those that need intubation and sedation are at an increased risk of ocular complications.[34] The most common direct ocular complications with SBI and smoke inhalation injury includes exposure keratopathy and corneal ulceration. Less commonly, corneal burns can occur secondary to heat generated by the fire.[18,35] Serious globe burn injuries typically result in cornea edema imparting a clouded appearance to the cornea.[17,34] The eyes of every burn patient should be irrigated liberally with saline and a thorough ophthalmological examination, including the use of fluorescein stain, should be performed.[18]

Indirect ocular injury can occur secondary to orbital compartment syndrome.[4,11,34,36,37] Orbital compartment syndrome results from progressive periorbital tissue swelling and eyelid contracture resulting in increased intraocular pressure and subsequent optic neuropathy and blindness.[34,36] Approximately 40% of severely burned human patients require a lateral canthotomy due to increased intraorbital pressure.[36] Intraocular pressures are ideally monitored daily for the first 72 hours postinjury.[34,36]

Veterinary patients presenting with evidence of smoke inhalation injury and SBI should be evaluated for paw pad burns. The same depth classification described for skin burns also applies to paw pad burns.[38] Paw pads contain a thicker epidermal layer that may provide some initial protection to the underlying germinal layers.[38] Like other burn wounds, time is required to determine the extent of the burn injury. All paw pad burns should be treated with daily debridement, application of topical antimicrobial medications, and bandaging.[38–40] During debridement, any viable pad tissue should be conserved.[41] Superficial wounds will heal via epithelialization and contracture, whereas deeper wounds may require reconstruction and paw pad transposition.[38,39] Paw pad injuries often take considerable time to heal due to the nature of the tissue and environmental stresses.[39]

Extremities must be evaluated for fractures and joint instabilities from trauma before resuscitation-associated edema develops. Progressive edema can cause profound limb ischemia secondary to swelling under circumferential eschars or within inelastic muscle compartments leading to extremity compartment syndrome.[11] It may be necessary to perform an escharotomy or fasciotomy in burned and unburned limbs if ischemia is noted.[17]

Immediate Treatment

Initial treatment includes establishing an airway via intubation if necessary. Immediate supplemental oxygen therapy is essential in animals with suspected smoke exposure and CM or cyanide toxicity.[18] Both CM and cyanide toxicities typically respond to oxygen therapy alone.[17] Supplemental oxygen administration improves SpO_2 and decreases the half-life of CO-Hgb.[6,28,42] The elimination half-life of CM is 5 hours at 21% oxygen, 1 hour at 100% oxygen, and approximately 20 minutes at 100% hyperbaric oxygen therapy (HBOT) at 2.5–3.0 atmosphere absolute.[6,28,31,32] Cyanide is rapidly metabolized making more specific treatments such as sodium thiosulfate and nitrites (amyl nitrite and sodium nitrite) uneccessary.[17] Oxygen may be delivered via a face mask, nasal cannula, oxygen hood, oxygen cage, or via intubation. Nasopharyngeal burns can hinder oxygen supplementation via nasal cannula.

Dogs evaluated for smoke exposure with subsequent neurologic and respiratory abnormalities typically improve 5–30 minutes after the institution of oxygen therapy.[25] Persistently hypoxic patients that fail to respond to supportive care may require mechanical ventilation or HBOT.[13,17–19,24,43,44] Specific ventilatory strategies for patients with smoke inhalation injury are beyond the scope of this review but are discussed in detail elsewhere.[44] Patients that present comatose or with significant neurologic signs are at greater risk for delayed neurologic sequelae (DNS).[27,28,32] Delayed neurological sequelae is characterized by a relapse in neurologic dysfunction after a transient period of improvement.[28,42] Delayed neurological sequelae is discussed in greater detail in the accompanying review.[1]

The use of HBOT in people with smoke inhalation injury and SBI is controversial.[28] A trend toward a decreased risk of DNS in patients treated with HBOT exists.[17,28] Hyperbaric oxygen therapy involves administering 100% oxygen in a pressurized chamber which accelerates the dissociation of CM from hemoglobin molecules.[28,32] In human medicine, HBOT is considered for hemodynamically stable patients who have a history of loss of consciousness, neurologic sequelae, or an ongoing metabolic acidosis.[17,24,28] Hyperbaric oxygen therapy is not entirely without risk with potential complications including pneumothorax, gas embolism, barotrauma, oxygen toxicity-induced seizures, pulmonary edema and hemorrhage, and oxidative stress.[17,28] As HBOT is not commonly available to veterinary patients the authors recommend administration of 100% atmospheric oxygen therapy to patients that lack a response to conventional supplemental oxygen therapy.

Initial venous access is obtained peripherally, ideally through unburned skin. To prevent ongoing tissue damage, dermal decontamination can be attempted to remove debris and toxin-laden soot from the patient, being careful to avoid excessively stressing the patient.[18] Ideally, a complete blood count, serum biochemistry, coagulation profile, arterial blood gas, plasma lactate, and CO-Hgb concentration (if available) are evaluated.

Cooling

Rapidly cooling burn wounds prior to arrival or shortly after arrival to the hospital is beneficial. Cooling burn wounds within 30 minutes of thermal injury prevents ongoing tissue damage, reduces edema formation, increases the speed of re-epithelialization, and improves cosmetic appearance.[45,46] Cooling the burn wound contributes to the analgesic regimen and improves wound healing by preventing progressive cellular necrosis that contributes to the zone of coagulation and zone of stasis.[45,47] The temperature of the water used to cool the burn wound has been extensively evaluated in people and animals models.[45–48] Current recommended first-aid treatment includes cooling the wounds with cold tap water (15°C [59°F]) for 20 minutes.[45] Colder water temperatures (2°C [35°F]) are beneficial but not as readily available as tap water.[45] Application of continuous running tap water decreases the depth of burn injury and improves wound healing when compared to the utilization of wet compress towels.[46] The utilization of ice water for cooling burns is controversial due to the lack of evidence for improved wound outcome.[48] Ice water cooling also increases the risk of hypothermia. The current recommendation is to avoid ice when cooling burn wounds.[45–48] The temperature of the patient should be monitored closely while cooling burn wounds to avoid hypothermia.

Fluid therapy

Fluid resuscitation is the single most important treatment for severe burn patients. The goal of fluid resuscitation is to maintain organ perfusion and avoid tissue ischemia with the least amount of fluid required. Within the first 1–2 hour after injury, burn patients experience little change in intravascular volume or hemodynamics. However, a delay in fluid resuscitation beyond 2 hours of burn injury reportedly complicates resuscitation and increases mortality.[11] After the initial 1–2 hour window, hemodynamic instability ensues for approximately 24–48 hours after SBI despite fluid resuscitation. Neither preload nor cardiac output is able to be normalized with fluid resuscitation until 24 hours after injury.[12] In SBI patients with concurrent inhalation injury, a marked increase in hemodynamic instability and a 30–50% increase in initial fluid requirement is seen when compared to patients with burn injury alone.[43,49]

Isotonic crystalloids

Patients with SBI are initially resuscitated with isotonic crystalloid solutions. There are a number of formulas that estimate fluid requirements for people with SBI.[49,50] The "Consensus formula" (formerly referred to as the "Parkland formula") has become the most widely used resuscitation guideline and is utilized to calculate the volume of crystalloid required within the first 24 hours after SBI.[11] The formula recommends administering 4 mL/kg per percentage TBSA in the first 24 hours, with half of this amount administered in the first 8 hours.[11–13,50] The remainder of the calculated fluid volume is administered over the remaining 16 hours.[49] Recent studies, however, have found that average volumes administered to contemporary burn patients significantly surpass formula predictions, often exceeding 6–7 mL/kg percentage TBSA.[11,12,17,49,50]

The current tendency to overuse crystalloid fluids in SBI patients has been coined "fluid creep."[49] Fluid creep is thought to result from increased utilization of crystalloids, excessive initial fluid resuscitation, prejudice against the use of colloids, lack of attention to fluid volumes by clinicians, and inaccurate estimation of TBSA.[49] Potential consequences of over-resuscitation include pulmonary edema, myocardial edema, conversion of superficial to deep burns secondary to edema formation, extremity compartment syndrome with need for fasciotomies in unburned limbs, massive pleural and pericardial effusion, and abdominal compartment syndrome.[11,49,51] The authors recommend applying the 'Consensus formula' as a guideline for resuscitation of small animals with SBI with a reduction of the calculated fluid volume by 25–50% for cats.[13] Fluid therapy should be titrated to maintain mean arterial pressure (MAP) and urinary output.

Natural and Synthetic Colloids

The use of colloids, both natural (eg, albumin) and synthetic (eg, dextrans and hydroxyethyl starches [HES]), in resuscitation of burn patients is controversial. Due to the concern for leakage of proteins and large molecules through compromised capillary membranes, it is recommended to wait at least 8–12 hours postburn injury before utilizing colloids.[12,21,51] However, in the presence of hemodynamic instability, the use of colloids in the first 24 hours may be necessary.[51] The use of colloids augments colloid osmotic pressure (COP) which has been documented to reduce edema formation in non-burned tissue (but not in the burn wound itself).[12] Colloid osmotic pressure, measured via a colloid osmometer, can be utilized to help guide colloid therapy.[52] Various artifacts including hemolysis and type of liquid anticoagulants used for blood sampling can confound results.[52] Therefore, the authors caution against the use of COP as the sole means to guide fluid therapy choices.

The use of human albumin (HA) in resuscitation of people with SBI remains contentious due the concern for increased risk of mortality and the lack of a clear benefit.[50,51,53] The 1999 Cochrane review noted a 6% mortality increase in patients receiving HA for burns, hypovolemia, or hypoproteinemia.[54] Since then, numerous meta-analyses and clinical trials have been performed in nonburn patients and have not found either a benefit nor an increased risk of mortality with the use of HA.[55–58]

Human albumin has been administered to critically ill dogs and cats for numerous disease processes.[59–62] The administration of HA results in an increase in serum albumin concentration, total plasma protein concentration, systolic blood pressure, and COP.[60,61] The administration of HA can result in immediate hypersensitivity reactions that can resolve with discontinuation of product administration.[61–63] Evaluation of 25% HA administration in healthy dogs revealed that both immediate and delayed hypersensitivity reactions can occur secondary to the development of anti-HA antibodies.[63] The use of HA should be considered only after a benefit-to-risk ratio is assessed.[59]

Lyophylized canine-specific albumin (CSA) solutions have recently been developed in the United States. Administration of CSA to both healthy dogs and dogs with septic peritonitis have been reported to increase serum albumin concentrations, systolic blood pressure, and result in variable increases in COP.[a,b,c] Canine-specific albumin appears to be well tolerated with no immediate or delayed hypersensitivity reactions being reported. In the absence of clinical trials evaluating CSA in critically ill animals and the lack of a clear benefit of HA in people with burns, albumin products should be utilized cautiously in small animals with SBI.

The utilization of synthetic colloids (eg, HES) remains a controversial and frequently debated component of fluid resuscitation in burn patients.[49] The use of high molecular weight HES preparations in the early postburn period leads to a marked reduction in fluid requirements and edema formation in people.[64,65] Infusion of low molecular weight, less substituted HES in people leads to improvement in hemodynamics and systemic oxygen delivery.[66] Concerns regarding their adverse effects, particularly on coagulation, have limited their use in burn resuscitation.[50] Newer low molecular weight HES appear to impair coagulation to a lesser degree when compared with older high molecular weight HES preparations.[67] However, all artificial colloids can potentially increase bleeding tendency after infusion of very large volumes.[68] Some burn centers administer colloids to patients who develop increasing fluid requirements during resuscitation, as a means of "escape" from fluid creep.[49]

Hypertonic saline

The use of hypertonic saline (HTS) is controversial in the resuscitation of human burn victims.[37,50,51,53] Administration of HTS creates a hyperosmolarity that results in plasma volume expansion due to the shift of fluid into the vascular space.[50,53] Fluid may also be mobilized from the interstitial space by osmotic action which may limit burn edema.[50] People resuscitated with HTS typically require less fluid in the first 24 hours to achieve target urine output than those receiving an isotonic crystalloid alone.[69,70] In addition, these patients have a decreased risk of abdominal compartment syndrome.[71] However, there is a 4-fold increase in the risk of kidney failure and an increase risk in mortality when HTS is utilized.[71]

Administration of large quantifies of HTS leads to hypernatremia. Hypernatremia of 160 mmol/L (160 mEq/L), corresponding to an osmolarity of 340 mOsm/L, results in a dramatic decline in urine production.[72] Hypernatremia and hyperosmolarity can result in brain shrinkage, intracranial vessel detachment, kidney failure, fluid overload, cerebral edema, and seizures.[53] The American Burn Association (ABA) guidelines recommend that the use of HTS should be reserved for experienced burn physicians, with close monitoring of plasma sodium concentrations.[12]

Monitoring resuscitation

Each patient responds uniquely to burn injury. Resuscitation strategies depend on age, extent, and depth of burn injury, concurrent inhalation injury and preexisting disease processes.[53] There is no single optimal parameter that reliably predicts adequate resuscitation and global perfusion. Traditional resuscitation parameters, such as urine production, heart rate, and arterial blood pressure are routinely monitored in tandem, although they do not precisely reflect global perfusion, regional microcirculation, or reversal of shock.[73] The limitations of traditional end point parameters have led to investigation of more invasive monitoring including pulmonary artery occlusion pressure, central venous pressure, intrathoracic blood volume, esophageal doppler monitoring of cardiac function, and right ventricular end-diastolic volume index.[50,53] The use of these hemodynamic monitoring tools often result in excessive fluid administration in an attempt to restore cardiac output and correct hypovolemia.[51] According to the ABA guidelines, preload-driven strategies for burn resuscitation are not advisable because neither preload or cardiac output restoration is achievable within the first 24 hours.[12] Invasive monitoring with central venous catheters or pulmonary artery catheters may be indicated only in patients with an inadequate response to standard treatment.[12]

Urine output is routinely measured in patients with SBI to guide fluid therapy and resuscitation.[11,50,53] The ABA guidelines recommend titrating fluids to maintain a urine output of 0.4 mL/kg per hour in adults and 1.0 mL/kg per hour in pediatric patients.[12] Due to potential complications of fluid overload and excessive edema formation, most experienced burn clinicians are accepting lower urine production as a resuscitation endpoint.[50] The authors recommend maintaining urine production between 0.5–1 mL/kg per hour in small animals with SBI if all other parameters are within reference intervals.

Standard endpoints of resuscitation in human burn victims include maintaining MAP > 70 mm Hg with a normal heart rate.[53] Despite appropriate fluid resuscitation, persistent tachycardia may be noted secondary to pain and anxiety making heart rate an unreliable indicator of hypovolemia.[11,50] Blood pressure should be monitored frequently.[11] Noninvasive blood pressure monitoring may be inaccurate in the face of tissue edema.[50,53] In addition, catecholamine-associated arteriospasm can falsely increase direct arterial pressure readings.[53] Despite the limitations to these parameters, it is best to monitor trends in both heart rate and blood pressure in small animals with SBI.

Plasma lactate and base deficit have been investigated in human burn patients to evaluate adequacy of resuscitation but the results have been controversial. An early study determined that despite appropriate MAP and urine output in resuscitated burn patients, both plasma lactate and base deficit remained increased.[74] This finding raised concerns that patients with adequate MAP and urine output may still be hypoperfused. Further studies have indicated that the failure to correct both plasma lactate and base deficit within 24 hours of burn injury was associated with increased mortality.[73–78] In addition, a persistently increased base deficit (< –6 mmol/L) after adequate resuscitation is associated with an increased incidence of ARDS, systemic inflammatory response syndrome, and multiple organ dysfunction.[79] However, several burn experts maintain that current burn shock resuscitation guidelines are appropriate and that these abnormalities in plasma lactate and base deficit do not indicate poor resuscitation, but are simply a reflection of burn size.[73,75] Concerns remain that attempts to normalize both plasma lactate and base deficit could lead to overzealous fluid administration and exacerbation of edema.[12] Currently, there is a lack of prospective studies supporting the use of these parameters to guide fluid resuscitation.

Wound Management

Burn wound care involves daily decontamination, debridement, and dressing of the wound.[4,9,11,13,21] On admission and once to twice daily thereafter, burn wounds should be cleaned with a 1:40 dilution of chlorhexidine or 1:9 dilution of povidone iodine solution to gently debride nonviable tissue.[2,4,5,9,11,13,21] Hydrotherapy is commonly utilized in human medicine for cleansing and debridément of burn wounds. Previously, immersion hydrotherapy was employed by most burn facilities, but due to fecal and nosocomial contamination of wounds this practice has largely been discontinued.[11,80] Patients are now showered with a hand-held sprayer, which reduces the risk of transferring surface bacteria to open burn wounds.[80]

Specific recommendations for management of wounds depend on the depth and severity of the burn in-

jury. Superficial burns heal within 1 week if the wound is kept moist and clean.[21] Superficial partial-thickness wounds heal within 1–2 weeks with simple cleaning and wound debridément.[4,5] Due to increased risk of infection, large blisters should remain intact for no more than two days.[5] Both deep partial-thickness and full-thickness burns require eschar removal and application of topical antimicrobial agents until wound closure or graft application is possible.[4,5] Surgical excision and grafting is recommended for burn wounds that are estimated to take longer than 2–3 weeks to heal with conservative management.[3,4,81] If these wounds are allowed to heal conservatively, patients experience longer hospital stays, accentuated pain, functional impairment, and significant hypertrophic scarring.[2–34,13,21,47,82,83]

Management of extensive burn wounds were previously more conservative than current recommendations. In the past, it was recommended that eschars naturally separate from healthy tissue with daily trimming of the freed edges.[80,81] Small wounds healed by re-epithelialization whereas larger wounds healed via formation of a granulation bed and subsequent skin grafting.[3,81] The natural healing conservative technique is recommended for smaller, less extensive burn wounds or for patients that are not stable enough for more immediate, radical surgical excision of devitalized tissue.

Early excision and grafting

Research has indicated that an earlier and more aggressive surgical approach leads to attenuation of the hypermetabolic response and decreased infection rates.[2,4,13,21,83–85] Early burn wound excision decreases release of proinflammatory mediators such as IL-1, IL-6, and TNF-α.[85–87] The increased permeability of the burn eschar causes excessive fluid, protein, immunoglobulin, and electrolyte loss.[4] In addition, the eschar promotes bacterial growth. Escharectomy is the best means of preventing bacterial infections and sepsis and exposes a viable bed of tissue for skin grafting or permanent wound closure.[4,88]

After initial stabilization, staged excision can be initiated as early as the third day post injury if feasible.[83,88] Surgeries can be spaced 2–3 days apart until the eschar is removed and wound closure is achieved.[81] Two different surgical techniques have been described for wound excision in human medicine: tangential excision and fascial excision[3,4,81,88] (Table 1). Detailed description of excision and graft techniques are beyond the scope of this article and the reader is referred elsewhere.[4]

Surgical excision, both tangential and fascial approaches, can result in blood loss although major bleeding is rare.[4,21,65,81] Multiple techniques have been described to reduce intraoperative blood loss including the application of topical epinephrine with or without thrombin and the use of tourniquets.[4,81,88] Epinephrine (1:10,000) soaked nonadherent dressings can be applied to the burn injury.[81] Another method, the "tumescent technique," involves infiltration of dilute epinephrine in 37°C (98.6° F) saline until the tissue becomes distended and has a smooth, firm texture.[88,89] This technique reduces total blood loss and transfusion requirements by 50% in burn patients undergoing tangential excision.[90] An alternate technique utilizes a tourniquet. Tourniquets can serve as a primary means of hemostasis for extremity burn wounds but application should be limited to <120 minutes to prevent ischemic injury.[4,81] Assessment of tissue viability can be hampered with this technique due to the lack of capillary bleeding.

Vacuum-assisted closure (VAC) has been utilized to treat severe burn wounds in both people and veterinary patients.[4,90,91] Vacuum-assisted closure applies topical negative pressure to a wound which improves dermal blood flow, removes edema, decreases bacterial colonization, and promotes formation of granulation tissue.[5,90,91] Vacuum-assisted closure also prevents the conversion of partial-thickness burns to full-thickness burns.[5] Vacuum-assisted closure was recently reported to successfully treat a dog with 50% TBSA SBI.[91]

Topical Treatment

Topical antimicrobial agents should be instituted after initial decontamination to prevent bacterial colonization of the burn wound.[6,17,90] Systemic agents are less successful in treating local infections because they do not reach the burn wounds in large concentration due to microthombosis of vessels and wound edema causing compression of the vessels that supply the area.[90] An antimicrobial agent is applied directly to the burn wound. The area is then covered with a nonadherent dressing. Multiple topical agents are available for treatment of burn wounds[2–6,9,11,13,17,80,90,92–98] (Table 2).

Silver sulfadiazine (SSD), a water-soluble cream base synthesized from silver nitrate and sodium sulfadiazine, is the gold standard in topical burn treatment.[2,3,5,9,11,13,17,80,90] Silver sulfadiazine has a broad antimicrobial spectrum, fair to good eschar penetration, and minimal adverse side effects.[6,9,17,90,92] Recently, sustained silver releasing products have been developed combining a silver agent with a "carrier" dressing. Such products can be applied to partial-thickness burns and can remain in place for 3–7 days.[3,6,17] This eliminates manipulation of the burn site and the pain associated with dressing changes.[3,11,90,92] Sustained release products are ideal for treatment of local burn injuries on an outpatient basis.

Mafenide acetate, another topical product, is typically used if patients develop antimicrobial resistance to silver products.[3] Mafenide acetate is a potent carbonic anhydrase inhibitor that has excellent antibacterial and bacteriostatic activity and excellent eschar penetration.[6,90,99] Potential side effects include pain on application and a reversible hyperchloremic metabolic acidosis when used on extensive burns.[3,5,6,17,90]

The use of honey for treatment of wounds has been employed for many years due to its antimicrobial properties, but limited information is available regarding its utility in burn wounds.[96,97] Antimicrobial properties include a high osmolarity, a low pH, and the production of hydrogen peroxide.[94,96,98,100] Nonstandardized honey, from the natural environment, has a variable antimicrobial spectrum whereas a medical-grade honey has a broad spectrum of action that is highly reproducible.[94] Honey not only provides a physical barrier to invading organisms and acts as a nonadherent dressing but it also provides a moist environment for wound healing.[95,98] Honey results in a greater healing rate, less contracture, decreased hypergranulation, increased wound strength, and a more sterile environment when compared to SSD.[95,97] Further studies are needed to evaluate the use of honey in the management of SBI.

The authors recommend applying SSD to all burn wounds for initial topical therapy. If delayed healing is noted or if there is evidence of a resistant infection, the clinician should switch topical therapy to either mafenide acetate or medical-grade honey. Other potential dressings for burn wounds include temporary and permanent skin substitutes, biosynthetic dressings, and synthetic dressings. Details of the various dressings are discussed elsewhere.[3–5,90]

Pain Management

Pain management is a topic of ongoing research and a continual challenge in the care of burn victims. Pain at the burn site is associated with dressing changes, tissue debridément, application of topical antimicrobial agents, hydrotherapy, physical therapy, and skin grafting.[101–103] No correlation has been found between

Table 1: Surgical excision

Type of excision	Technique	Utility	Advantages	Disadvantages
Tangential	Shave layers of devitalized tissue off until exposing a viable tissue bed (capillary bleeding noted)	Small burns	More cosmetic procedure Preserves body contours	More blood loss Longer procedure time
Fascial	Complete excision of skin, subcutaneous fat, involved muscle fascia	Large, deep life-threatening burns	Faster procedure time Less skin grafting Less blood loss	Severe cosmetic deformity Loss of cutaneous nerves

Table 2: Topical medications: spectrum of action, advantages, disadvantages

Topical agent	Spectrum of action	Advantages	Disadvantages
Silver sulfadiazine (SSD)	Broad spectrum Most gram positive Most gram negative MRSA Yeast Molds	Painless, soothing Penetrates eschar No metabolic side effects Sustained release products available	Possible delayed wound healing Delays eschar separation Argyria Local hypersensitivity reaction
Mafenide acetate	Broad spectrum Most gram positive Most gram negative Minimal anti-fungal Limited against staphylococci (MRSA)	Excellent eschar penetration Effective against SSD resistant bacteria	Painful on application Local hypersensitivity reaction Reversible hyperchloremic acidosis
Honey	Broad spectrum Most gram positive Most gram negative MRSA	Improved healing rate Less wound contracture Decreased hypergranulation Increased wound strength Creates more sterile environment Provides physical barrier to wound	Local hypersensitivity reaction Tissue dehydration

Table 3: Etiology and characteristics of burn pain

Type of pain	Pain ptimulus	Characteristics
Procedural pain	Debridement	Burning and stinging with sharp pains
	Dressing changes	Persists for minutes to hours after dressing changes
	Hydrotherapy	
	Physical therapy	
Background pain	Occurs at rest	Prolonged duration
	Normal daily activity	Relatively constant nature
		Continuous "burning" or "throbbing"
		Mild to moderate intensity
Breakthrough pain	Movement after long period of immobility	Occurs despite stable analgesic regimen
		Severe intensity
		Short duration
		Unpredictable

pain scores and burn size, burn depth, or the number of days elapsed since burn injury.[102–104] Wound closure significantly dampens the intensity of burn pain but long-term pain after wound closure occurs in 52% of burn victims.[17,103]

Burn pain can be classified as procedural pain, background pain, or breakthrough pain based on the stimulus and characteristics of the pain[101–103] (Table 3). The intensity of burn pain fluctuates from day to day and patients should be evaluated and reassessed continuously to adjust analgesic regimens.[101,104] Each category of burn pain should be evaluated individually for optimal analgesia.

A multimodal analgesic regimen is most effective for management of pain.[101–103] During the acute phase of burn injury, intravenous opioids should be the primary method of analgesia.[101,103] In people, opioid doses often significantly exceed standard dosing recommendations and can escalate rapidly during burn treatment secondary to opiate tolerance and increased clearance during the hyperdynamic hypermetabolic phase.[11,101,103] Procedural pain is associated with significant anxiety which may increase over time.[104] In people, anxiety associated with the impending therapeutic procedure can markedly enhance the degree of perceived pain.[101–103] It is unknown if this phenomenon occurs in veterinary patients. Benzodiazepines decrease background and procedural pain and should be used in conjunction with an opioid during burn treatment.[11,101,102] Sedative hypnotics such as ketamine and propofol have been shown to significantly reduce pain and anxiety associated with procedural pain.[11,101,102,d]

Assessment of pain in veterinary medicine can be challenging. Physiologic parameters such as heart rate, blood pressure, and respiratory rate are considered the least accurate when assessing for pain in human patients.[101] Behavioral pain indicators have been assessed in both people and animals.[101] In people able to communicate after burn injury, pain assessment tools including "0 to 10" numeric rating scale, visual analog, descriptor words (eg, adjectives), face, and color scales are frequently utilized.[101,102] The Glasgow Composite Measure Pain Scale is a behavior-based composite scale that is used to assess pain in small animals and could be considered for use in burn patients.[105–107]

Nutrition

During the hypermetabolic phase, burn patients experience increased muscle catabolism and a negative nitrogen balance resulting in the loss of lean body mass and severe muscle wasting. Accurate caloric and protein requirement estimation and appropriate supplementation are necessary to support metabolic energy requirements.[85] Institution of enteral feeding within 24–48 hours postburn injury is recommended.[2,11,13,17,85] Enteral nutrition is superior to parenteral nutrition because it maintains gut motility, decreases plasma endotoxin and inflammatory mediators, preserves "first-pass" nutrient delivery to the liver, and decreases intestinal ischemia and reperfusion injury.[108] Parenteral nutrition should be administered only when patients do not tolerate enteral nutrition due to vomiting, oral ulceration, prolonged ileus, or during the perioperative period.[11,17,108]

Anti-oxidants

Anti-oxidant therapies have been evaluated with respect to their ability to limit production of reactive oxygen species in burn patients.[12,109,110] High-dose vitamin C has demonstrated the most promising results by reducing postburn lipid peroxidation, edema of burn and non-burned tissue, and vascular permeability.[111] Vitamin C infusion in experimentally burned sheep resulted in a 30–45% reduction in fluid requirements.[110,111] Despite the need for prospective clinical trials, the ABA guidelines currently recommend the use of high-dose vitamin C.[111] Experimentally, high-dose vitamin C administration (14–66 mL/kg per h) within the first 24 hours of SBI in dogs results in reduced lipid peroxidation and postburn microvascular leakage.[111–113]

Complications

Infection

Burn patients are at high risk for developing wound infections, pneumonia, and sepsis secondary to the loss of the natural cutaneous barrier, necrosis of the

endobronchial epithelium, and the use of invasive devices.[11,13,16,17,80,114] The incidence of burn wound infections has dramatically decreased with the institution of early burn excision and skin grafting.[90] The hyperdynamic hypermetabolic response to SBI and chronic stimulation from inflammatory mediators causes patients to be persistently tachycardic, tachypneic, have a leukocytosis, and a baseline temperature reset 2°C (3.6°F) higher than a nonburned patient. These physiologic responses to SBI make all of these criteria unreliable indicators of infection or sepsis.[11,85,114] In burn patients, the presence of SIRS criteria is not a specific indicator of wound infection.[114]

Diagnosis of burn wound infection or sepsis is based on clinical signs and examination of the wound.[17,80,114] Wounds should be monitored frequently to evaluate for local infection.[4,17,80,114] Signs of infection include change in wound color, increased exudate, increased pain, increased wound depth, or early separation of eschars.[115] The clinical diagnosis of wound infections can be supported by a quantitative tissue biopsy.[17,80] Wound infection is defined as a bacterial count exceeding 10^5 microorganisms per gram of tissue.[5,80,115] A positive swab culture does not indicate wound infection since skin and wounds are normally colonized with bacteria characterized by a bacterial count less than 10^5 microorgansims per gram of tissue.[5,80,115] When quantitative cultures yield $< 10^5$ microorganisms per gram of burn wound tissue, it has been found that 96.1% of patients do not have an actual burn wound infection.[80] A quantitative tissue biopsy culture is useful to determine the predominant microorganism in the wound and to provide susceptibility data to guide antimicrobial selection.[80] Clinical indications of a systemic infection in people include alterations in mental status, worsening pulmonary function, impaired renal function, inability to tolerate enteral feedings, and persistent hyperglycemia despite aggressive insulin therapy.[114,116] The clinical diagnosis of sepsis can be supported via cultures of blood, urine, and sputum.[17,80]

Approximately 50–70% of patients with SBI and concomitant smoke inhalation injury develop pneumonia.[11,16,17] Patients with smoke inhalation injury can also develop ARDS which complicates the diagnosis and treatment of pneumonia.[13,14,19,26] Pneumonia should be suspected with radiographic evidence of new pulmonary infiltrates and signs of sepsis.[114] Confirmation of pneumonia via transtracheal wash or bronchoalveolar lavage is recommended.[18,114]

Systemic antimicrobials are not administered prophylactically in the management of burn wounds.[2] Topical antimicrobial agents are efficient in preventing and treating local burn wound infections in the absence of systemic signs of sepsis.[90,117] Systemic antimicrobials should be administered for 1 week when a local burn wound infection is present with a concomitant systemic infection.[114,117] In this circumstance, antimicrobial infusion under the burn wound as well as aggressive debridement is recommended.[114] Antimicrobial selection should be based on culture and susceptibility results. Plasma peak and trough concentrations of antimicrobial should be monitored to determine the appropriate dosing regimen due to altered pharmacokinetic parameters that occur after burn injury.[13,117] Perioperative antimicrobial administration is indicated to prevent surgically induced bacteremia or endotoxemia.[13,117] However, the prophylactic use of antimicrobials during dressing changes is not necessary.[117]

Hypothermia

Hypothermia is a common complication in SBI patients. Cooling of burn wounds, large-volume fluid resuscitation, low ambient temperatures, topical wound treatments, and prolonged surgical procedures all contribute to hypothermia.[81,118] Hypothermia can have profound adverse effects on coagulation, drug metabolism, oxygen consumption, and risk of infection.[11,118] Attempts to maintain normal body temperature in burn patients include warming the room to 33°C (91.4°F) to decrease evaporative losses, warming inspired air, utilizing warming blankets and countercurrent heat exchangers for infused fluids.[10,80,84] This increase in ambient temperature also augments the hypermetabolic response.[13,85]

Intra-abdominal Hypertension

Intra-abdominal hypertension (IAH) and secondary abdominal compartment syndrome (ACS) are common sequelae to SBI in people.[119–121] The occurrence of IAH and secondary ACS as a result of SBI is unknown in veterinary medicine. Intra-abdominal pressure (IAP) can be altered by decreased abdominal wall compliance resulting from circumferential torso burns and tension secondary to pain or discomfort.[119] As IAP increases, IAH ensues. If IAH is unnoticed or left untreated, ACS develops resulting in pressure-induced organ dysfunction and failure.[119] IAH and ACS occur in 36–70% and 1–20%, respectively, in people with SBI.[122]

In veterinary medicine, IAP has been evaluated in healthy dogs and cats.[123–125] It is unknown what pressure constitutes IAH in dogs and cats, therefore it is recommended that pressures > 10 cm H_2O be considered mild elevations in IAP.[123] In the veterinary literature, it is recommended that therapeutic intervention should be instituted with IAP > 20 cm H_2O in dogs and cats.[123]

In people, continuous intra-bladder pressure is recommended to be monitored in patients with >30% TBSA burns[11] or in SBI patients that receive >250 mL/kg

Table 4: Noninvasive therapy for intra-abdominal hypertension

Improve compliance
Sedation and analgesia to decrease thoracoabdominal muscle tone
Perform escharotomy on circumferential torso burns
Alter gastrointestinal motility and volume
Prokinetics (eg, erythromycin or metoclopramide)
Nasogastric drainage
Enemas
Evacuate intraperitoneal fluid
Percutaneous catheter abdominal decompression

of crystalloids in 24 hours due to an increased risk of ACS.[11,120,126] If the estimated crystalloid requirement (eg, via the use of the Consensus formula) exceeds the threshold (>250–300 mL/kg per day), then utilization of colloids during resuscitation should be considered.[122,127] If IAH is noted, noninvasive therapy must be attempted. (Table 4).[11,120–122] If the IAH is refractory to these treatments, decompressive laparotomy with delayed closure is indicated.[11,121]

Prognosis

Over the last 2 decades, the survival rate after SBI and smoke inhalation injury has steadily improved with advances in burn care. The overall mortality rate in human burn victims without inhalation injury is approximately 14% compared to a mortality rate of 20% in burn victims with concomitant inhalation injury.[128,129] Prognostic factors that increase the likelihood of mortality in human burn victims include increased TBSA involvement, concomitant inhalation injury, and increasing age.[128] The development of pneumonia after smoke inhalation injury is also associated with an increased risk of mortality.[16]

The overall mortality in veterinary patients with burn injury is unknown. Based on human data, animals with SBI and concomitant lung injury are likely to have a higher risk of mortality than those with burn injury or lung injury alone.[18] Dogs and cats suffering from smoke inhalation injury alone are reported to have a 90% survival rate.[18,25,26] Progression of respiratory dysfunction in dogs and cats on the second day of hospitalization appears to indicate an increased morbidity with an increase in length of hospitalization.[25,26] More prospective studies are needed to evaluate prognosis in small animal patients wih burn injury and smoke inhalation.

Conclusion

Local burn injuries can be treated conservatively and are unlikely to lead to systemic derangements. Severe burn injury leads to significant hemodynamic instability, massive fluid shifts, and hypovolemia requiring prompt and aggressive therapy. Patients with SBI likely have concomitant smoke inhalation injury that may require supplemental oxygen therapy. Burn wounds initially should be cooled for 20 minutes with continuous running tap water to prevent progressive tissue damage. Daily wound management including debridement and application of an antimicrobial agent covered with a nonadherent dressing is recommended. Early excision and grafting significantly decreases the hypermetabolic response and risk of infection associated with SBI. Patients should be monitored closely for complications including hypothermia, infections, and IAH. Without early and aggressive intervention following SBI, burn shock ensues, rapidly leading to organ failure and death. Due to the limited number of domestic animals treated for SBI, the majority of the recommendations are based on advances in human medicine. Future studies in veterinary medicine are warranted.

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Footnotes

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